and speculative discussion which tends to surround the "beta blockade withdrawal syndrome", confining our conclusions to the specific agent and clinical setting tested. If it can ever be considered reasonable to withdraw a beta blocker then based on our data in patients with severe stable angina it should surely not be considered a "dangerous" practice to stop treatment with atenolol in patients who have mild or no symptoms?

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# Effect of timolol on changes in serum potassium concentration during acute myocardial infarction

Sir,

Nordrehaug *et al* (1985; **53:** 388–93) showed that the administration of timolol after myocardial infarction reduces the frequency of hypokalaemia during the first 24 hours after the infarct. For greater accuracy they should have used plasma rather than serum because there is erratic leakage from erythrocytes during coagulation.

Using insulin-induced hypoglycaemia (in healthy volunteers) as another model for acute stress, we have also observed that prior non-specific beta blockade with nadolol or propranolol prevents hypokalaemia.[1 2] Since these effects of stress are mediated through a pronounced increase in plasma catecholamine concentrations it is pertinent to mention that adrenaline-induced influx of potassium into leucocytes in vitro is inhibited by the non-selective beta blocker, timolol and that these cells are probably a model for body cells as a whole.[3] In addition, beta blockade in the hypoglycaemia model[1] reduces the magnitude of (*a*) increase in serum free fatty acid concentrations by inhibiting lipolysis; (*b*) the increase in various haemostatic variables like factor VIII related antigen; (*c*) platelet aggregation. These effects are related to the pathogenesis of myocardial infarction since they are all arrhythmogenic or prothrombotic.[4–6] It is also important to determine whether selective blockade of $beta_1$ receptors produces similar results since some of the above effects of catecholamines are thought to be mainly mediated by $beta_2$ receptors.

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